Information

Hypotension and the Shock Syndrome in Myocardial Infarction

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Material Supplied by the California Heart Association

HYPOTENSION IS A COMMON and often benign consequence of myocardial infarction while the "shock syndrome" is a less common, but often fatal complication. Precise definition and clear understanding of the hemodynamic consequences of myocardial infarction are, therefore, necessary for management.

Blood pressure alone is not a good index of the patient's clinical status. Blood pressure (BP) is a function of both cardiac output (CO) and total peripheral resistance (TPR) as indicated by the simple relationship:

$$BP = CO \times TPR$$

Thus, BP will be reduced if either CO or TPR is reduced.

Shillingford and colleagues have shown that there are two physiological patterns associated with arterial hypotension depending upon the state of peripheral resistance. In one group of patients, the hypotension is clearly related to a reduced cardiac output and the peripheral resistance may be normal or increased. In the second group, TPR is reduced but the cardiac output is normal. These two groups can sometimes be distinguished clinically. The first group with increased resistance presents with cool extremities and a small pulse volume while the second group is characterized by warm extremities and a full pulse.

Hypotension exists in at least 80 percent of patients following myocardial infarction and in most patients the blood pressure will return to normal levels with the relief of pain and the administration of oxygen. The hypotension occasionally persists for weeks or months, but is often unassociated with significant symptoms.

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The Shock Syndrome

The shock syndrome occurs in about 20 percent of patients with myocardial infarction and accounts for at least 50 percent of the deaths now that the mortality from arrhythmias has been reduced. The mortality rate in patients with the shock syndrome secondary to myocardial infarction ranges from 85 to 95 percent if the syndrome is rigidly defined and clearly distinguished from simple hypotension as described above.

The following criteria for the shock syndrome define a population of patients with a mortality of greater than 95 percent. (1) Systolic arterial blood pressure of less than 80 mm Hg, (2) Clinical signs of peripheral circulatory insufficiency; cold, moist skin and cyanosis, (3) Dulled sensorium, (4) Oliguria with urine flow of less than 30 ml/hr, and (5) Failure of improvement to follow relief of pain and the administration of oxygen.

The insult to the heart is the cause of the shock syndrome in myocardial infarction although all organ systems are ultimately involved. The function of the heart is impaired by the initial insult and this results in a decrease in arterial pressure and, hence, coronary blood flow because of its dependence upon aortic perfusion pressure. The reduction in coronary perfusion pressure and myocardial blood flow further impair myocardial function and may increase the size of the myocardial infarction. Arrhythmias and metabolic acidosis also participate in this deterioration in that they are the result of inadequate perfusion and both tend to perpetuate the precipitating conditions. It is this negative feedback relationship (impaired cardiac function—arterial hypotension —reduced coronary blood flow—impaired cardiac function) which accounts for the high mortality associated with the shock syndrome.

Cardiac output is lower in a population of patients with shock than in those who do not have the shock syndrome, but this is by no means the whole explanation. There are many patients with myocardial infarction without shock who have cardiac output in the same range or lower than that measured in patients with shock and, therefore, it is not possible to characterize these patients on the basis of changes of cardiac output alone.

Total peripheral resistance, the other factor important in determining blood pressure, may be either normal increased or decreased in myocardial infarction. Here again, a similar range of values for total peripheral resistance can be seen in pa-

tients in the absence of shock. Normally, a fall in cardiac output is accompanied by a compensatory rise in total peripheral resistance, but in patients with shock due to myocardial infarction the appropriate response in peripheral resistance fails to occur. Thus, it appears that the total peripheral resistance is inadequate to support blood pressure at the existing level of cardiac output, regardless of the extent of reduction of the latter.

Treatment

The objective of treatment is the interruption of the negative feedback loop whereby impaired myocardial function leads to a reduction in arterial pressure, decreased coronary blood flow and a further depression of left ventricular function. This objective is approached by attempting to improve cardiac function and to raise the arterial blood pressure.

Vasopressors constitute an important form of therapy for shock of myocardial infarction. A small increase in arterial pressure may result in a sizable increase in coronary blood flow. The best vasopressors for use in myocardial infarction are norepinephrine (Levophed®) and metaraminol (Aramine®) which act both on the alpha receptors in the arterial wall and also on the beta receptors in the myocardium. Thus, the practical experience with the treatment of shock in myocardial infarction is consistent with the theory of pathogenesis which emphasizes the dual nature of the pathophysiology in that drugs which act on both the heart and the peripheral circulation are the most effective.

Consideration of the central role of impaired myocardial function in the shock syndrome leads to the conclusion that cardiac glycosides should be administered to all patients with this condition. Obviously, the cardiac glycosides cannot improve the function of necrotic myocardium, but a positive inotropic influence of the non-infarcted myocardium is desirable. It has been demonstrated that the incidence of arrhythmia and cardiac rupture is no higher in patients with myocardial infarction treated with digitalis than in a control group.

Certain general measures have proven useful in the treatment of the shock syndrome. All patients with the shock syndrome should receive 100 percent oxygen continuously because the addition of dissolved oxygen to the plasma helps to combat the hypoxemia which is universally present. The relief of pain is important as some vasodepressor reflex activity may be a response to severe pain, but narcotics should be used cautiously in view of their hemodynamic effects. Fluid volume replacement has a limited, but definite, place in the therapy of the shock syndrome due to myocardial infarction. It may be indicated in patients who have been receiving pressor drugs for a prolonged period because pressor therapy results in a decrease in plasma volume secondary to the movement of fluid into extravascular space. In such patients, if central venous pressure is low and there is no evidence of pulmonary congestion, the blood pressure may be easier to maintain after plasma volume has been expanded by the administration of plasma or salt poor albumin. Venous pressure should be monitored and the lungs examined frequently during the administration of plasma. Also, fluid replacement is necessary in patients who have lost extracellular fluid volume consequent to vomiting or sweating.

The high mortality and relative ineffectiveness of conventional therapy has provided the stimulus for the investigation of other approaches to the problem. The basic defect in the shock syndrome is impaired myocardial function and, therefore, many mechanical assist devices are currently under investigation. The therapeutic value of hyperbaric oxygen therapy is also under study. Studies with experimental animals are encouraging, but clinical trials have been disappointing. A large fraction of patients with the shock syndrome have severe, diffuse coronary atherosclerosis with large areas of infarcted myocardium. It is in this group of patients that total replacement of the heart by a homotransplant or an artificial device will have its greatest potential usefulness. Circulatory assist devices may have their greatest use in sustaining life until this is possible.